



Parasitological and pathological findings of coccidiosis in an experimental infection caused by *Eimeria ahsata* in lambs

Nader Ahmadi Saleh Baberi^a, Iraj Karimi^b, Hossein Nourani^c, Hamidreza Azizi Alavije^b,
Gholamreza Razmi^c

^a Graduate student of the Veterinary Parasitology, Faculty of Veterinary Medicine, Ferdowsi University of Mashhad, Mashhad, Iran.

^b Department of Pathobiology, Faculty of Veterinary Medicine, Shahrood University, Shahrood, Iran.

^c Department of Pathobiology, Faculty of Veterinary Medicine, Ferdowsi University of Mashhad, Mashhad, Iran.

ABSTRACT

This study was conducted to investigate the pathogenesis process of *E. ahsata* and its morphological, pathological, and distribution of lesions in the involved tissues during 42 days of infection. Twelve lambs were randomly divided into two groups including the control and the infected groups after confirmation of their health. The animals in the experiment group were orally infected with 1×10^5 sporulated oocysts. From 7 days after inoculation (DAI), the feces were sampled and oocysts per gram of feces (OPG) were individually examined for each lamb. At 7, 14, 21, 28, 35, and 42 DAI, one lamb from each group was necropsied and the abomasum, small and large intestine, mesenteric lymph nodes, spleens, and livers were grossly investigated. From 21 to 42 DAI, mild to severe clinical lesions such as congestion and edema were seen on the mucosal surface of the small intestine associated with white and small foci about 1-2 mm, especially jejunum and ileum. From 7 DAI to the end of the study various stages of the parasite life cycle, infiltration of inflammatory cells, epithelial hyperplasia of villi, and destruction of villi epithelium were seen. The results showed that *E. ahsata* was pathogenic in lambs and the macro and microscopic lesions were mostly seen in the jejunum.

Keywords

Eimeria ahsata; Sheep; Pathology; OPG

Number of Figures: **6**
Number of Tables: **2**
Number of References: **30**
Number of Pages: **9**

Abbreviations

OPG: oocysts per gram

DPI: days post inoculation

DAI: days after inoculation

FS: fecal score

Introduction

Coccidiosis is one of the most common parasitic diseases, caused by the genus *Eimeria* spp. in the alimentary system of sheep in the world [1,2]. The disease is more common in lambs aged 4-6 months [3]. The rearing stressor conditions such as weaning, transportation or transfer to a new pen, malnutrition, overcrowded population, and unsuitable weather play an important role in the incidence of the disease in sheep and goats [4]. The genus *Eimeria* causes the death of a large number of host intestinal cells and enterocytes leading to reduced absorbance of the critical electrolytes and nutrients [5]. The most common clinical signs of the disease are diarrhea, weight loss, anemia, rough hair coat, and weakness [5]. In addition, the disease reduces the production of meat and milk products and increases the mortality rate. The mechanism and grading of tissue damage depend on *Eimeria* species, the number of oocysts ingested, stress condition, age, physical condition, genetic sensitivity, and host immune system. Due to the sensitivity of young animals, the clinical form of the disease is reported at this age [6].

In the small ruminant, the induced hyperplasia by coccidia results in the thickness of the intestinal wall leading to poor absorbance of nutrients, diarrhea, and dehydration [4]. The mild to moderate histopathologic changes can be associated with the thickness of intestinal mucosa as well as the formation of the plaque or the nodules with 1-2 mm in diameters in size [4]. In some *Eimeria* species, large schizonts are considerably seen. The most common clinical lesions of coccidiosis in young sheep and goats are non-pedunculated whitish nodules on the intestinal mucosa. These plaques are adhered to each other in advanced infection [4]. To date, 12 intestinal and 1 abomasal *Eimeria* species including *E. crandallis*, *E. bakuensis*, *E. weybridgeensis*, *E. ovinoidalis*, *E. intricate*, *E. gilruthi*, *E. pallid*, *E. faurei*, *E. parva*, *E. marsica*, *E. granulose*, *E. bakuensis*, and *E. ahsata* have been identified in sheep [3].

Among the above species, *E. ovinoidalis*, *E. ahsata*, *E. crandallis*, and *E. bakuensis* are considered serious pathogens [3]. Regarding high prevalence of *E. ahsata* in sheep of different areas of Iran [7,8,9], this experimental study was conducted to study the pathogenesis of this parasite and to evaluate the morpho-pathology and distribution of lesions in lambs.

Results**Clinical signs**

There were no clinical signs till 20 DAI in all animals. Diarrhea was the first clinical sign that was observed in two lambs at 21 DAI, which led to anorexia, weakness, dehydration, mucosal paleness and weight

loss. The fecal samples consistency of these lambs were semiliquid (Fecal score=2) at 21 DAI and watery diarrhea (fecal score= 3) at 35 DAI (Table 1). No clinical signs were seen in other lambs in infected and control groups and fecal samples consistency were normal till the end of experiment.

OPG rate

The level of oocysts per gram of feces (OPG) of each lamb after the pre-patent period that was varying from 7 to 42 DAI are shown in Table 1. Two lambs with diarrhea had high OPG.

Body weight

Based on the results of the present study, the lambs in the case group had less weight gain than the control group and in the studied model, a significant difference was observed between the two groups ($p < 0.05$) (Table. 2).

Histopathological findings

Gross lesions. There was no considerable gross lesion in the sacrificed lambs at 7 and 14 DAI. Mild to severe lesions were seen in the jejunum and with less intensity in the ileum at 21 DAI. The congestion and edematous state of small intestine mucosa, especially, in the jejunum and ileum were identified. The small intestinal mucosal thickness associated with congestion and white nodules with 1-2 mm in diameter were detected on the internal surface of mucosal jejunum and ileum at 28 DAI. In addition to white nodules were creased on the small intestinal mucosal in particular jejunum at day 35 of infection (Figure 1).

At 42 DAI, the advance and diffuse glandular lesions associated with mucosal thickness and creasing from serosal surface of jejunum with less intensity in the ileum were noted (Figure 2). There was no gross lesion in the abomasum, liver, and spleen of all animals. The enlargement of mesenteric lymph nodes was the commonly detected lesion on all infected animals at 21, 28, 35, and 42 DAI. No gross lesions were observed in the gastrointestinal tract in lambs of the control group from the beginning to the end of the study.

Microscopic lesions. At 7 DAI, vascular congestion of mucosal and submucosal surfaces associated with different stages of the parasite life cycle including micro and macrogametes, and schizonts were seen in the jejunum and ileum. The epithelial and lymph tissues hyperplasia, infiltration of inflammatory cells such as eosinophilic cells in the lamina propria and villous tip as well as denuded villous tip and hyperplasia were seen in the ileum and jejunum.

At 14 DAI, the various stages of the parasite life

Table 1.

The changes of OPG and fecal score in each lamb of infected group during experimental infection with *E. ahsata* at a dose of 1×10^5

Lamb	7 DAI		14 DAI		21 DAI		28 DAI		35 DAI		42 DAI	
	OPG	FS	OPG	FS	OPG	FS	OPG	FS	OPG	FS	OPG	FS
1	0	1	-	-	-	-	-	-	-	-	-	-
2	0	1	1200	1	-	-	-	-	-	-	-	-
3	0	1	1000	1	38,000	2	-	-	-	-	-	-
4	0	1	800	1	15,000	1	8400	1	-	-	-	-
5	0	1	1000	1	33,000	2	30,000	3	45,000	3	-	-
6	0	1	1000	1	10,000	1	6400	1	8000	1	3000	1

OPG: Oocyst per gram

DAI: Day(s) after infection

FS: Fecal score

cycle were seen in more parts of the small intestine. The vascular congestion of mucosal and submucosal, epithelial and lymph tissues hyperplasia, infiltration of inflammatory cells, and eosinophilic infiltration in the lamina propria and villous tip as well as denuded villous tip and hyperplasia was seen in the small intestine. The second-generation schizont and merozoites were detected within villi epithelial cells of the small intestine. Other stages of parasite life cycle such as progamonts, the developed micro and macrogametes, and a few oocysts were also detected within villi epithelial and crypts of duodenum, jejunum, and ileum. Most lesions were observed in the jejunum. A few first-generation schizonts were seen in the livers, spleens, and mesenteric lymph nodes.

At 21 DAI, microscopic intestinal lesions were widely detected. Infiltration of lymphocytes and eosinophils in lamina propria with less intensity in small intestine submucosa. The various forms of the parasite including gamonts and a large number of oocysts were seen in different parts of the small intestine in particular the jejunum (Figure 3). The infiltration of lymphocytes was also seen in the ileum. The round micro containing a large number of basophilic and clear nuclei as well as the round macro-gametocytes containing a large number of eosinophilic granules were seen. There was no lesion in the cecum, colon, liver, spleen, and mesenteric lymph nodes.

At 35 DAI, The microscopic lesions were widely detected in the ileum and jejunum and some cases in the colon. The losses of epithelial surface, infiltration of eosinophils, micro and macrogametes and oocysts associated with lymphatic hyperplasia, mucosal thickness resulting from papillary hyperplasia, and infiltration of inflammatory cells especially eosinophils in the lamia propria were seen (Figure 4). Microscopically, nodular hyperplasia and noted white pulp were seen in the spleen. The liver and mesenteric lymph nodes were reported as normal and hyperplastic particularly in the cortex, respectively.

At 42 DAI, congested livers, spleen, and mesenteric lymph nodes were reported. In the jejunum, losses of the integrity of the lieberkühn gland, infiltration of inflammatory cells, micro and macrogametes, and the developed oocysts were seen. In the ileum, the presence of oocysts, villous epithelial hyperplasia, losses of integrity, infiltration of eosinophils, plasma cells, macrophages, micro, and macrogametes was seen (Figure 5). There was no lesion in the abomasum, rectum, and colon.

Table 2.

Comparison of the body weight (Kg) of lambs in the infected and control groups

DPI	Weight (Kg)	
	Control group	Test group
0	23.2 ± 0.87	23.60 ± 1.2
7	23.3 ± 0.62	23.7 ± 1.1
14	23.2 ± 0.53	22.32 ± 0.93
21	$24.65 \pm 0.85^*$	$21.62 \pm 1.13^*$
28	$25.7 \pm 1.04^*$	$21.66 \pm 1.09^*$
35	27.2 ± 1.30	-
42	27.3 ± 1.32	-

* $p < 0.05$ was accepted as statistically significant.

DPI: days post inoculation



Figure 1.
The whitish nodules (arrows) on mucosal surface of the jejunum at 35 DAI



Figure 2.
Cerebriform or gyrate pattern and depressions on the serosal surface of jejunum (arrows) at 42 DAI.

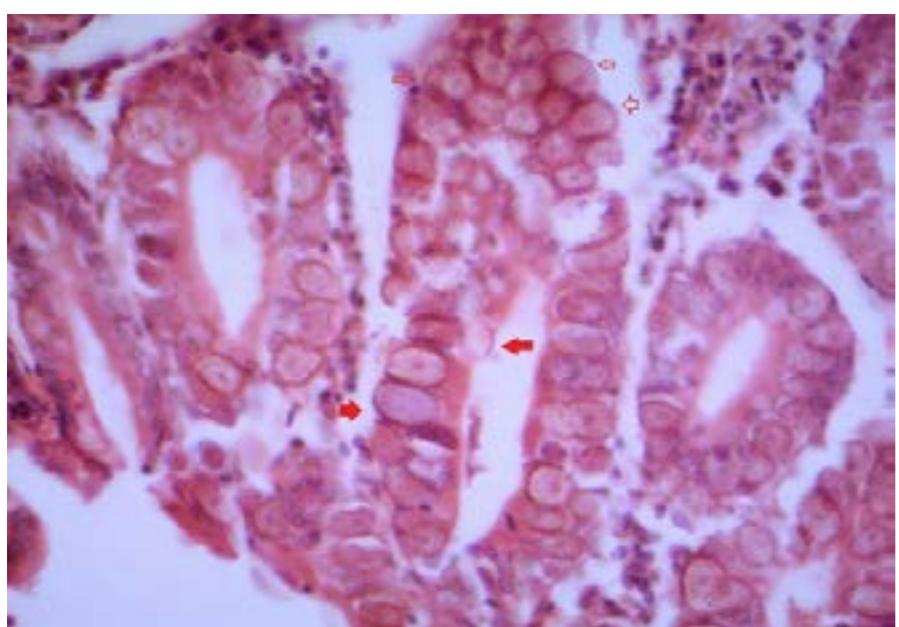


Figure 3
Histopathological section of jejunum at 21 DAI. There are a large number of gamonts (hollow arrows), and oocysts (solid arrows) within the epithelial tissue of intestinal glands. H&E, $\times 400$

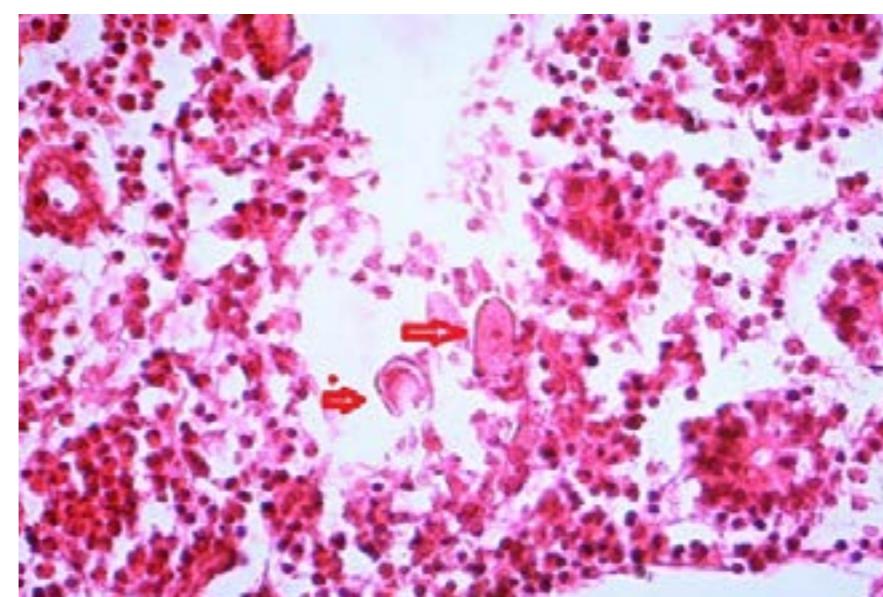


Figure 4
Histopathological section of jejunum at 35 DAI. The presence of oocysts (arrows) associated with infiltration of eosinophils and other inflammatory cells, creating villi. H&E, $\times 400$

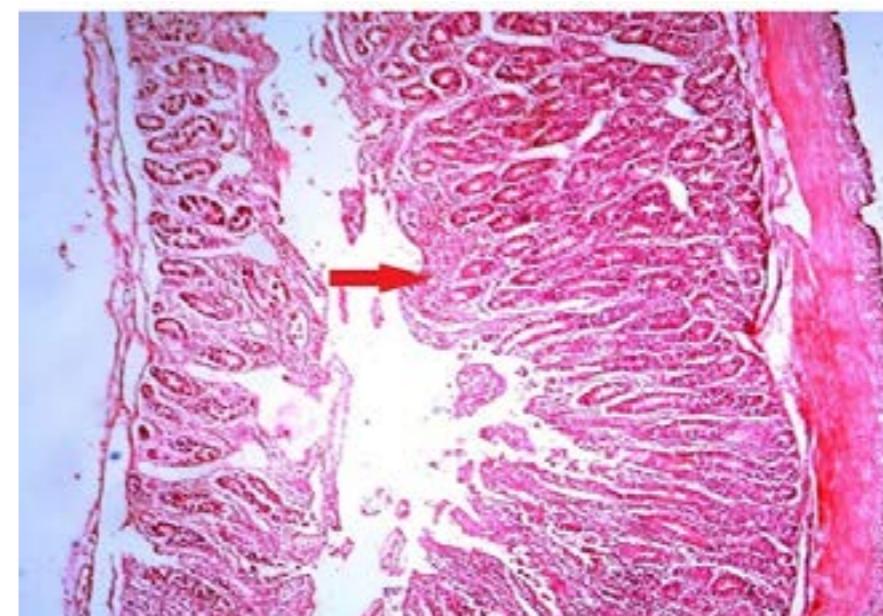


Figure 5.
Histology of the jejunum at 42 DAI with the presence of oocysts, infiltration of eosinophils and other inflammatory cells, associated with partial epithelial hyperplasia and hyperemia (arrow). H&E, $\times 100$

Discussion

Eimeria ahsata has been known as the most common Eimeria species in the sheep in Iran, Spain, and China [8,15,5]. In the present study, the pathogenicity of *E. ahsata* as one of the common species in lambs was experimentally investigated. The first detection of oocysts was at 14 DAI in fecal samples of the infected group which are consistent with the prepatent period of *E. ahsata* about 12 to 18 days in an experimental study [16]. Clinical signs appeared with diarrhea, anorexia, dehydration, and weakness in two infected lambs at 21 DAI. Few studies have been performed on the pathogenicity of *E. ahsata*. Smith et al

(1960)

showed that oral occultation with 1×105 *E. ahsata* oocysts caused diarrhea, loss of appetite, and listlessness at 15-16 DAI and death in some lambs at 23-32 DAI [17]. Mahart and Sherrick (1965) showed the low pathogenicity of *E. ahsata* in feedlot lambs [18]. Cathpole et al (1976) compared the pathogenesis of four Eimeria species in lambs for 4 weeks. They reported no clinical signs in the lambs when *E. ahsata* oocysts were given 10 to 1000 oocysts per day in a week, whereas *E. ovinoidalis* caused diarrhea in lambs. The difference in clinical signs severity in experimental studies may be related to infective dose, age, sheep breed, infective dose, and concomitant in-

jor role in the destruction of villi and crypts, with the release of histamine from damaged intestinal cells in parasitic infection. Other studies have been reported an ileoileal intussusception associated with proliferative [26]. The mild to severe hyperplasia in the crypt and villi of the small and large intestine in naturally occurring coccidiosis in sheep [26]. In addition, necrosis, denuding of villi and intestinal gland epithelium, congestion, infiltration of inflammatory cells associated with various stages of *Eimeria* such as micro and macrogamete and oocysts in the small intestinal mucosa has been also reported in infected lambs [27]. For comparison, similar microscopic lesions such as hyperplasia of epithelial cells of villi and crypts of jejunum and ileum, remarkable infiltration of lymphocytes and eosinophils have been reported in kids that experimentally infected by *E. arloingi* [22].

It seems that the tissue damage intensity depends on the *Eimeria* species, the number of the inoculated oocysts, host immune system, age genetic, nutrition, and stress [28,29].

The gain weight in the present study was significantly decreased in the infected group compared to the control group. The loss of body weight associated with clinical coccidiosis is mainly due to loss of nutrients as a result of parasite-induced mucosal lesions and, to a lesser degree, to alterations of intestinal digestion and absorption of nutrients [30]. Subclinical coccidiosis may also lead to reduced growth, uneven lamb size, and a higher food conversion ratio [31].

Materials & Methods

Isolation of *E. ahsata*

Before the start of the experiment, The fecal samples of 70 referred lambs with diarrhea from the School of Veterinary Medicine were examined by the Mac-Master method [10]. A portion of each positive sample (3 gram) was mixed in 42 ml of phosphate buffer solution (PBS) and filtered through the sieve (Azman co. Iran) to omit the large particles. The filtered suspension was centrifuged at 2000 rpm for 5 min and the sediment was mixed with 2.5% (w/v) aqueous potassium dichromate solution (1:5) in Petri dishes and kept in a climate chamber. It was aerated continuously for twenty days at 27 °C. The rate of sporulated oocysts was determined by microscopic examination when more than 90% of oocysts were sporulated, and they were stored at 4°C until used. The frequency of the different *Eimeria* species with special regard to the pathogenic species was determined based on the morphological characteristics of the oocysts and related to the OPG counts [10,11]. Fifty-three samples were positive for *Eimeria* spp. The *E. ahsata* was the most prevalent (79%) species and other species included *E. ovinoidalis* (1%), *E. bakuensis* (1%), *E. granulosa* (2%), *E. crandalis* (7%), *E. faurei* (18%), and *E. intricata* (25%) less prevalent in the fecal samples of diarrheic sheep. The fecal samples containing 95 -100 % *E. ahsata* and more than 500 OPG were chosen for the experiment [11]. *E. ahsata* oocyst was ovoid with non-round polar cap, yellowish-brown, no residual body, and large steady body with sporocyst residuum, 33.4 × 22.6 µm

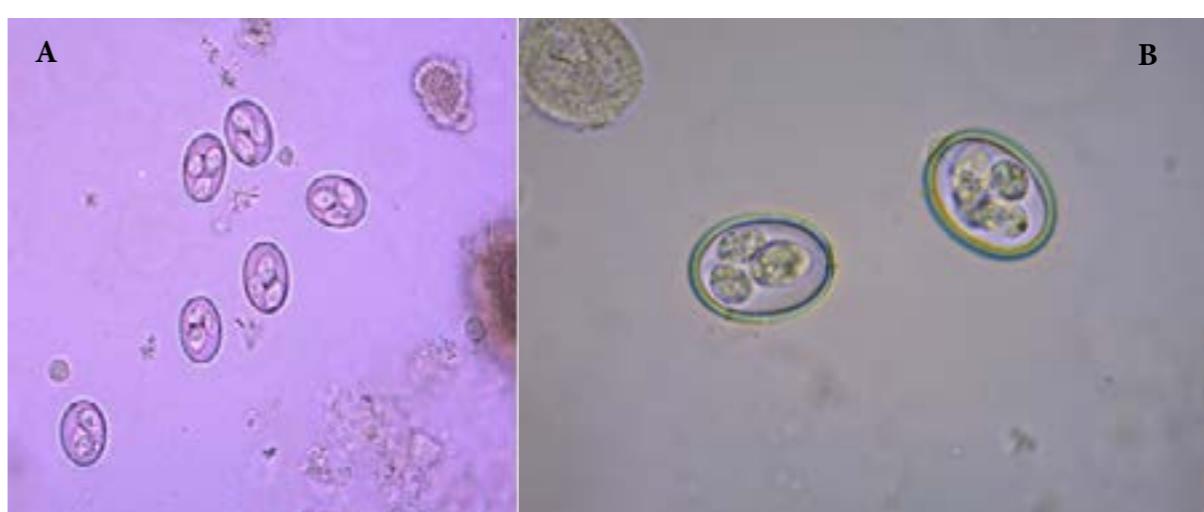


Figure 6.
A. Sporulated *E. ahsata* oocysts, $\times 100$. B. Sporulated *E. ahsata* oocyst, $\times 400$

(Figure 6).

Experimental examination

Twelve female lambs (*Ovis aries*), 2 months old were obtained from a non-infected herd raised under hygienic conditions. The lambs were transferred to the Research Center in the Faculty of Veterinary Medicine, Ferdowsi University of Mashhad. After clinical examination and confirmation of their health, the lambs were placed in individual pens in a protective environment for three weeks, to adapt to the diet and new environment. Lambs were fed daily with a standard diet consisting of alfalfa hay and concentrate during the study period. In addition, the fecal samples of lambs were examined three times a week to ensure not to have coccidia. Thereafter, the coccidia-free lambs were randomly divided into two equal infected and control groups. Before inoculation of the lambs, the oocytes suspension was washed with PBS solution by repeated centrifugation at 2000 rpm for 5 min until removal of potassium dichromate. Finally, the volume of the sediment was increased to 300 ml by adding distilled water, and the number of oocysts was calculated for each ml of suspension by Mac-Master methods. A single inoculum of an aqueous suspension 1×10^5 sporulated oocysts (50 mL) was given to each lamb with a stomach tube [12]. The lambs of the control group received distilled water (50 mL per animal). During the study, clinical signs and parasitological findings including anemia, diarrhea, body condition, and OPG of each animal were evaluated. The fecal sample of each lamb was collected directly from the rectum in the morning at 7, 14, 21, 28, 35, and 42 DAI. The score of consistency of feces was assessed as follows: Normal to pasty (1), semiliquid to liquid (2), watery (3), hemorrhagic, and/or with tissue (4) [13]. In addition, the number of oocysts per gram (OPG) was counted using the Mac-Master method. Body weights were assessed by weighting each lamb on a scale of kilograms at 7, 14, 21, 28, 35, and 42 DAI before euthanasia.

Necropsy and histopathology

At 7, 14, 21, 28, 35, and 42 DAI, a lamb from each group randomly was euthanized by intravenous sodium pentobarbital solution and underwent necropsy for clinical evaluation of elementary system [14]. For microscopic examination, the tissue samples from the duodenum, jejunum, ileum, cecum, colon, abomasum, mesenteric lymph nodes, liver, and spleen were harvested and fixed in 10 % formalin buffer (Merck, Germany). The samples

from small and large intestines were chosen with 10 cm intervals from the beginning of the duodenum to the end of the colon for microscopic examination. The fixed samples were embedded in paraffin, sectioned at 5 µm, and routinely stained using hematoxylin and eosin (H&E).

Statistical analysis

The SPSS software, version 22 (SPSS Inc., Chicago, USA) was used for data analysis. The student's *t*-test was used for investigating the effects of sampling time on the OPG and weight in two groups. *p*-values less than 0.05 were considered significant.

Ethics approval and consent to participate

The experiment on animals in the present study was approved by the Ethics Committee of Ferdowsi University of Mashhad (Approval ID: IR.UM.REC.1399.072).

Authors' Contributions

NASB: Methodology, Software, Formal analysis, investigation, Writing-Original draft preparation. IK: Supervision, Methodology, investigation, Resources, Writing- Reviewing and Editing. HN: Validation. HAA: Validation, Resources. GR: Supervision, Conceptualization, Visualization, Resources, Writing- Reviewing and Editing.

Acknowledgements

The research leading to these results was funded by a grant (No. 3.49852) from the Research Council of the Ferdowsi University of Mashhad, Iran

Competing Interests

The authors declare that they have no competing interests

References

- Molina JM, Ruiz A. Coccidiosis in sheep. In: J.P.Dubey (Eds) .Coccidiosis in livestock, poultry, companion animals and human. Taylor & Francis Group: CRC Press; 2017.
- Bangoura B, Bardsley KD. Ruminant Coccidiosis. Vet Clin North Am Food Anim Pract. 2020; 36: 187-203.
- Taylor MA, Coop RL, Wall RL. Veterinary Parasitology 3rd ed. Blackwell Publishing, 2007.
- Khodakaram-Tafti A, Hashemnia M. An overview of intestinal coccidiosis in sheep and goats. Revue Méd.Vét. 2017; 168: 1-3.
- Wang CR, Xiao JY, Chen AH , Chen J, Wang Y, Gao JF, Zhu XQ. Prevalence of coccidial infection in sheep and goats in northeastern China. Vet.Parasitol. 2010; 174: 213-217.
- Jolley WR, Bardsley KD. Ruminant coccidiosis. Vet. Clin. Food Anim. 2006; 22: 613-621.
- Heidari H. Identification of Eimeria species in sheep of Hamedan suburb, Iran J Vet Res. 2011; 66: 165-167.
- Hashemnia M, Rezaei F, Chalechale A, Kakaei S, Gheichivand S. Prevalence and intensity of Eimeria infection in sheep in western Iran. Int J Livest Res. 2014; 4:107-112.
- Mirzaei M, Dahmardeh E. The Prevalence of Eimeria Species in Sheep in Zabol city, Iran. Iran Vet J. 2016; 11:98-105.
- Eckert J, Taylor M, Catchpole J, D. Licois, P. Coudert, Bucklar H. Identification of Eimeria species and strains: Morphological characterization of oocysts. In:J. Eckert, R. Braun, M.W. Shirley, P. Coudert, P. (Eds) COST 89/820 Biotechnology—Guidelines on Techniques in Coccidiosis Research. European Commission Directorate-General XII, Science, Research and Development: Agriculture Biotechnology, Luxembourg, 1995. pp.103-119.
- Joachim A, Altreuther G, Bangoura B, Charles S, Daugschies A, Hinney B, Lindsay DS, Mundt HC, Ocak M, S. Sotiraki. W A A V P guideline for evaluating the efficacy of anticoccidials in mammals (pigs, dogs, cattle, and sheep). Vet .Parasitol. 2018; 253: 102-119.
- Gregory MW, Catchpole J. Ovine coccidiosis: the pathology of Eimeria crandallis infection. Inter.J.Parasitol.1978; 17:1099-1111.
- Bangoura B, Daugschies A. Parasitological and clinical parameters of experimental Eimeria zuernii infection in calves and influence on weight gain and haemogram. Parasitol Res. 2007; 100:1331-1340.
- Reilly JS. Euthanasia of Animals Used for Scientific Purposes. Anzccart press, 2001.
- Hidalgo-Argüello MR, Cordero-Del-Campillo M. Epizootiology of Eimeria ahsata coccidiosis in León (Spain). Vet.Parasitol. 1988; 27: 183-191.
- Catchpole J, Norton C, Joyner L. Experiments with defined multispecific coccidial infections in lambs. Parasitol. 1979; 72: 137-147.
- Smith MN, Davis LR, Bowman GW. The pathogenicity of Eimeria ahsata, a coccidium of Sheep. J.Protozool. 1960; 7: (Suppl.), 8.
- Mahart JL, Sherrick GW. Coccidiosis due to E.ahsata in feedlot lambs in Illinois. J. Am. Vet. Med. Ass. 1965; 146: 1415-1416.
- Chartier C, Paraud C. Coccidiosis due to Eimeria in sheep and goats, a review, Small Rumin. Res. 2012; 103: 84-92.
- Stockdale PHG, Bainborough AR, Bailey CB, Niilo LM. Some pathophysiological changes associated with infection of Eimeria zuernii in calves. Can J Comp Med.1981; 45: 34-37.
- Enemark HL, Dahl J, Enemark JM. Eimeriosis in Danish dairy calves--correlation between species, oocyst excretion and diarrhoea. Parasitol Res. 2013; 112: 169-176.
- Hashemnia M, Khodakaram-Tafti A, Razavi SM, Nazifi, S. Experimental caprine coccidiosis caused by Eimeria arloingi: morphopathologic and electron microscopic studies. Vet. Res.Com. 2012; 361:47-55.
- Khodakaram Tafti A, Mansourian M. Pathologic lesions of naturally occurring coccidiosis in sheep and goats. Comp Clin Pathol. 2008; 17: 87-91.
- Buret A, Gall DG, Nation PN, Olson ME. Intestinal protozoa and epithelial cell kinetics, structure and function. Parasitol Today.1990; 6:375-380.
- Aleksandersen M, Landsverk T, Gjerde B, Helle O. Scarcity of $\gamma\delta$ T Cells in intestinal epithelia containing coccidia despite general increase of epithelial lymphocytes. Vet .Pathol. 1995; 32:504-512.
- Khodakaram.A. Ileoileal intussusception associated with coccidiosis in sheep. B. J.Vet.Med. Series B, 1995;46 : 659-663.
- Taghavi-Razavizadeh A, Nourani H, Razmi G, Zarif MBH. A Report of unusual diffuse nodular lesions in intestines of Lambs with Coccidiosis. Vet ClinPathol.2016; 39: 251-254.
- Daugschies A, Bürger HJ, Akimaru M. Apparent digestibility of nutrients and nitrogen balance during experimental infection of calves with Eimeria bovis. Vet Parasitol.1998; 77: 93-102
- Jubb KVF, Kennedy PC, Palmer N. Pathology of domestic animals. Academic Press Inc,2007.
- Daugschies A, Najdrowski M. Eimeriosis in cattle: current understanding. J Vet Med Series B.2005; 52: 417-427.

Parasitological and pathological findings of coccidiosis

- de la Fuente C, Cuquerella M, Carrera L, Alunda JM. Effect of subclinical coccidiosis in kids on subsequent trichostrongylid infection after weaning. Vet Parasitol.1993; 45: 177-83.

COPYRIGHTS

©2021 The author(s). This is an open access article distributed under the terms of the Creative Commons Attribution (CC BY 4.0), which permits unrestricted use, distribution, and reproduction in any medium, as long as the original authors and source are cited. No permission is required from the authors or the publishers.



How to cite this article

Ahmadi Saleh Baberi N, Karimi I, Nourani H, Azizi Alavije HR, Razmi GR. Parasitological and pathological findings of coccidiosis in an experimental infection caused by *Eimeria ahsata* in lambs. Iran J Vet Sci Technol. (2021); 13(2): 20-28.
DOI: <https://doi.org/10.22067/ijvst.2021.71247.1057>
URL: https://ijvst.um.ac.ir/article_40441.html

Parasitological and pathological findings of coccidiosis